Alcohol's Effects on the Cardiovascular System

Over the last century, the medical and scientific communities have generally considered alcohol to be a toxin for the heart. Chronic heavy drinking is a leading cause of cardiovascular illnesses such as cardiomyopathy (degenerative disease of heart muscle), coronary artery disease, high blood pressure, dangerous heart rhythms (arrhythmias), and stroke.

However, as early as 1926, Raymond Pearl, one of the pioneers of modern epidemiology, noted that moderate drinkers had the longest life expectancy, followed by abstainers, then heavy drinkers (Pearl 1926). Some 70 years later, studies of mortality in widely disparate populations (Camargo et al. 1997; Keil et al. 1997; McElduff and Dobson 1997) have reported that moderate drinkers are 25 to 40 percent less likely to die from coronary heart disease (CHD) than abstainers are. An American Cancer Society prospective study (Boffetta and Garfinkel 1990) that followed more than 275,000 middle-aged men for 12 years found that men who consumed one drink daily had a lowered risk for CHD mortality (figure 1). Men who consumed three or more alcoholic drinks a day also had lower rates of CHD mortality compared with abstainers but increased rates of death from stroke, cancer, accidents, and violent crimes.

Alcohol in low to moderate amounts thus seems to have the potential for beneficial as well as toxic effects on the heart. The 1995 report of the Advisory Committee to the Secretaries of Health and Human Services and Agriculture on the *Dietary Guidelines for Americans* (U.S. Department of Agriculture [USDA] 1995*b*) acknowledges the evidence of an association between moderate drinking—defined in the guidelines as no more than two drinks a day for men and one drink a day for women—and lower risk of CHD in some groups. However, research has not confirmed that alcohol itself causes the lower risk. It is also plausible that the lower risk might result from some as yet unidentified factor or surrogate associated both with alcohol use and lower CHD risk, such as lifestyle, diet, exercise, or additives to alcoholic beverages (U.S. Department of Health and Human Services [USDHHS] 1999). This section highlights recent research examining the deleterious as well as the potentially beneficial effects of alcohol on the cardiovascular system, as well as the potential cellular mechanisms underlying these effects. An excellent summary of previous studies on the effects of alcohol on the heart can be found in the *Ninth Special Report to the U.S. Congress on Alcohol and Health* (National Institute on Alcohol Abuse and Alcoholism 1997).

The Heart

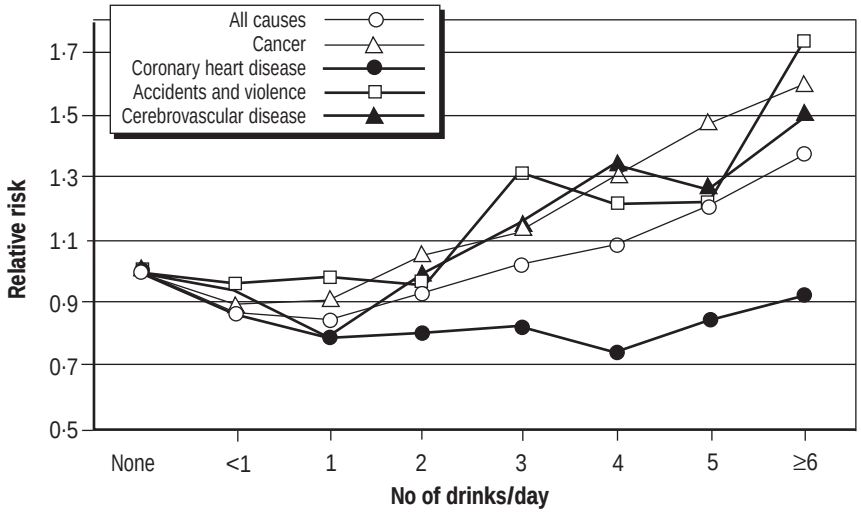
Alcoholic Cardiomyopathy

Long-term heavy drinking can cause the heart to become enlarged and lose some of its ability to contract, a condition known as alcoholic cardiomyopathy. This type of cardiomyopathy is nonischemic, meaning it is not the result of a loss of blood supply to the heart. Alcoholic heart disease is the most common cause of nonischemic cardiomyopathy in Western societies, responsible for up to 45 percent of cases (Kasper et al. 1994;

Definitions Related to Drinking

Studies investigating the health effects of alcohol vary in their definitions of "low," "moderate," and "heavy" drinking. According to the *Dietary Guidelines for Americans*, issued jointly by the U.S. Department of Agriculture and the U.S. Department of Health and Human Services, moderate drinking is no more than two standard drinks per day for men and no more than one per day for women (U.S. Department of Agriculture 1995). The National Institute on Alcohol Abuse and Alcoholism further recommends that people aged 65 and older limit their consumption of alcohol to one drink per day. Information on drinking levels as they are defined in the individual studies cited in this report can be found in the original references.

Figure 1: Alcohol consumption and relative risk of death over 12 years in American Cancer Society prospective study of 276,802 men aged 40–59



Mortality ratios are adjusted for age and smoking habits and for the four most common causes of death and death from all causes.

Source: Marmot and Brunner 1991. Reprinted with permission from *British Medical Journal*, Vol. 303, pp. 565–568, 1991. Copyright 1991, BMJ Publishing Group, London.

Sugrue et al. 1992). Alcoholic cardiomyopathy often leads to heart failure and death.

Most studies examining the prevalence of alcoholic cardiomyopathy have focused on male alcoholics. However, two recent studies found that women are more sensitive to alcohol's toxic effects on the heart and therefore have a greater risk than men of developing alcoholic cardiomyopathy (Fernandez-Sola et al. 1997; Urbano-Marquez et al. 1995). In both studies, alcoholic cardiomyopathy was as prevalent in female as male alcoholics, even though the mean lifetime dose of alcohol for women was only 60 percent that of male alcoholics. Why women are more sensitive to alcohol's toxic effects on the heart is unknown and should be a focus of future research (see the next section in this chapter, "Alcohol and Women: An Overview").

Interestingly, people survive longer with alcoholic cardiomyopathy than with other nonischemic cardiomyopathies, such as those caused by viral infection or pregnancy. A recent study showed that 81 percent of people with alcoholic cardiomyopathy were still alive after 5 years, while only 48 percent of people with other forms of cardiomyopathy survived that long despite comparable severity of symptoms and similar structural changes in the failing heart (Prazak et al. 1996).

Other researchers have found that alcoholic cardiomyopathy is totally or partially reversible with abstinence, unlike other nonischemic cardiomyopathies, in which irreversible heart damage and failure often occur (Francis et al. 1990). These findings suggest that disease progression is different in alcoholic cardiomyopathy than in other nonischemic cardiomyopathies. In a study of alcoholics with cardiomyopathy who were admitted to a detoxification unit, there was evidence that heart muscle damage was reduced or disappeared with abstention (Ballester et al. 1997). The researchers assessed damage to the heart muscle by measuring heart uptake of radioactively labeled antibodies to the muscle protein myosin. Over time, levels of

antimyosin antibodies decreased or disappeared, and cardiac ultrasound demonstrated that heart muscle function was improved. Alcoholics in the study with no evidence of heart disease did not have antimyosin antibody uptake even when they had been drinking a similar length of time and had similar total lifetime consumption as those with cardiomyopathy. This suggests that some people may be genetically predisposed to developing alcoholic cardiomyopathy.

Basic Mechanisms of Heart Muscle Damage

Researchers are exploring possible cellular and molecular mechanisms of alcohol's toxic effects on the heart, which may include alcohol-induced damage to the surface membrane of heart muscle cells, the myocytes; damage to important intracellular organelles and the apparatus that controls the cell's contractile machinery; or alteration of the myocyte's ability to synthesize proteins and enzymes. These alcohol-induced disruptions of myocyte integrity, contraction, or ability to self-repair can lead to cell death. Recent studies suggest at least two mediators of alcohol's toxic effects on myocytes: (1) production of oxygen-containing molecules, called reactive oxygen species (ROS), which damage cells (Husain and Somani 1997), and (2) detrimental changes in the receptors on the myocyte surface that regulate intracellular function (Strasser et al. 1996).

ROS, highly reactive molecular fragments produced as a by-product of alcohol metabolism, can cause serious harm to cells. Normally ROS are rapidly inactivated by antioxidants, but if the level of antioxidants is low or if ROS are over-produced, cell death can result. According to the ROS theory, chronic heavy alcohol consumption both increases the levels of ROS and decreases the levels of antioxidant enzymes that protect against cell damage by reducing ROS. Phospholipids, the backbone of cell membranes, are primary targets of the destructive process initiated by ROS, or peroxidation, and damage to the myocyte's membrane decreases cell integrity and intracellular functions, leading to cell death. Loss of myocytes, in turn, results in cardiomyopathy. A recent study of rats given large amounts of alcohol over long periods supports the ROS theory, finding that lipid peroxidation increased 149 percent, while levels of antioxidant enzyme activity decreased approximately 80 percent (Husain and Somani 1997).

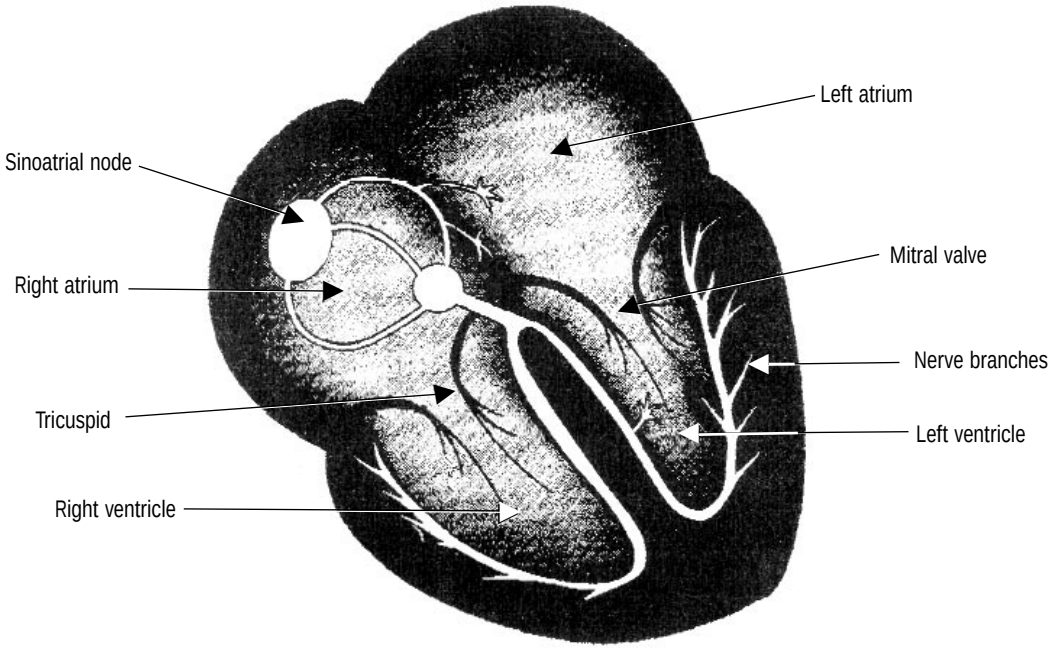
According to the myocyte surface receptor theory, alcohol causes harmful changes to signaling receptors—molecules on the cell surface that dock with signaling molecules outside the cell, causing corresponding changes inside—thereby damaging the ability of myocytes to maintain proper metabolism and contraction and leading to myocyte death and cardiomyopathy. A recent study in rats found that chronic alcohol consumption of up to 8 weeks reduced levels of two important classes of myocyte receptors, alpha-adrenergic receptors and muscarinic receptors (Strasser et al. 1996). When receptor activity is not normal, it can activate self-destructive mechanisms, leading to myocyte death. Future studies should focus on the intracellular consequences of alcohol's effects on increased ROS levels and myocyte receptor activity.

Arrhythmias

The heart's ability to function effectively as a pump depends on regular, synchronous contraction of the heart muscle (figure 2). Heavy drinking can disrupt the heart's own intrinsic pacemaker system and in this way increase the risk of abnormal changes in heart rhythm both acutely (during an episode of drinking) and chronically (due to long-term use).

The two most common types of irregularities, or arrhythmias, associated with heavy drinking are atrial fibrillation (rapid, irregular beating originating in the upper or atrial heart chambers) and life-threatening ventricular tachycardia (very rapid beating originating in the lower or ventricular chambers). Recent studies have aimed to identify the cellular mechanisms underlying alcohol's disruption of the heart's pacemaker system.

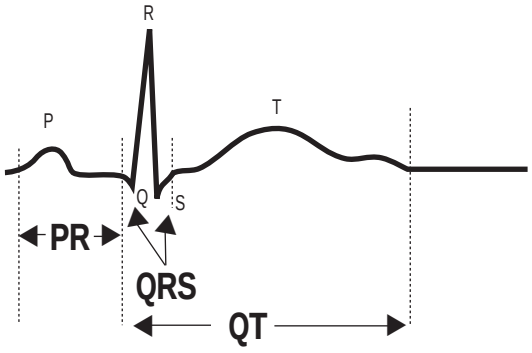
Figure 2: The heart's electrical system



The heart's electrical system stimulates the contraction of the muscle cells of the heart's four chambers, thereby causing blood to circulate through the heart chambers in a precise and sequential fashion. Electrical stimulation begins in the sinoatrial node, proceeds through the right and left atria, then moves through the ventricles.

Source: Chung and Rich 1990.

Figure 3: Sample electrocardiogram



The PR interval represents conduction from the sinoatrial node through the atria and into the atrioventricular chamber. The QT interval represents conduction through the ventricles and the return of conduction to baseline for the next heartbeat. Abnormalities in the segment between the S and T waves suggest heart injury.

Intoxication and Heart Rhythm

Intoxication can cause atrial fibrillation and other arrhythmias in both alcoholics and otherwise healthy persons. The development of arrhythmias with binge drinking—a condition seen most frequently around the holidays—is known as "holiday heart syndrome." Arrhythmias during intoxication result from alcohol-induced disturbances in the electrical discharge from individual myocytes and in the conduction of that electrical impulse through the heart muscle. A recent study demonstrated that in healthy intoxicated subjects, changes occurred in the electrocardiograms—surface recordings of the heart's electrical conduction (Cardy et al. 1996). In these subjects, the duration of electrical conduction through the atrial chamber of the heart—termed the P wave (figure 3)—was prolonged. A prolonged P wave increases the likelihood of developing atrial fibrillation.

Study of cellular transmission of electrical impulses provides clues to how alcohol disrupts the contraction of heart muscle. Electrical conduction through the heart muscle results when the movement of ions, including sodium, calcium, and potassium, across myocyte surface membranes changes the electrical potential of the

cells from negative relative to the exterior—the "resting" condition—to positive, a process called depolarization. This electrical impulse moves sequentially across myocytes in the heart muscle, resulting in synchronous contraction.

A recent study found that alcohol in concentrations within the range of what could be measured clinically in people who drink decreases calcium ion movement across myocyte membranes (Habuchi et al. 1995). This change could alter depolarization and conduction of the electrical impulse through the heart, leading to arrhythmias. These investigators also demonstrated that high doses of alcohol altered the movement of sodium ions across the myocyte membrane. Such changes could contribute to the increased incidence of death from arrhythmias among alcoholics who have engaged in binge drinking.

Alcohol Withdrawal and Heart Rhythm

Sudden death due to ventricular arrhythmias is one of the causes of mortality in alcoholics with or without preexisting heart disease (Wannamethee and Shaper 1992). Interestingly, blood alcohol levels in alcoholics who suffer sudden death due to cardiac causes often are low or undetectable (Clark 1988), suggesting that death occurred during abstinent periods. This finding supports the theory that sudden death due to cardiac causes in alcoholics may be related more to development of arrhythmias during alcohol withdrawal than to the toxic effects of alcohol during intoxication.

If the portion of the electrocardiogram called the QT interval (see figure 3) is prolonged, it signals delayed conduction and a predisposition to life-threatening ventricular arrhythmias. In a study of 62 alcoholics admitted for detoxification, researchers found that nearly half had a prolonged QT interval during withdrawal (Otero-Anton et al. 1997). When the symptoms of withdrawal were over, the QT interval returned to normal. Other researchers found higher than normal levels of adrenaline and noradrenaline among alcoholics undergoing detoxification (Denison et al. 1997). These hormones increase sensitivity to arrhythmias. Body stores of magnesium and potassium also were decreased, a change that can lengthen the QT interval and increase the chances of developing arrhythmias.

Heart ischemia—damage resulting from deficiency of blood flow due to narrowing or blockage of a vessel—also increases sensitivity for developing life-threatening arrhythmias. A recent study found that, during withdrawal, alcoholic men without clinical heart disease showed changes in the ST segment of the electrocardiogram, an indicator of ischemia (Denison et al. 1997). Twenty-four-hour continuous electrocardiograms revealed episodic ST segment depressions in more than a third of subjects. Such ST segment depressions in people with atherosclerotic heart disease reflect ischemia and are associated with increased risk of myocardial infarction—heart attack—and arrhythmia. This finding suggests that alcohol withdrawal should be considered a condition in which acute cardiac complications, such as ventricular arrhythmias, may be expected in susceptible alcoholics.

The Vascular System

Alcohol and Coronary Heart Disease

Detrimental Versus Beneficial Effects of Alcohol.

Heavy drinking increases the risk of heart attack due to CHD. Studies have shown that binge drinking increases episodes of angina (heart pain) in people with heart disease (Rossinen et al. 1996) and raises the risk of fatal heart attack (Kauhanen et al. 1997*a*; McElduff and Dobson 1997). Researchers in Finland recently reported that binge drinkers had six times the risk of fatal heart attack as moderate drinkers (Kauhanen et al. 1997*a*). In another study, this research group found that frequent hangovers were associated with a greater than twofold increase in death due to CHD (Kauhanen et al. 1997*b*). These studies underline the importance of preventive actions regarding not only the amount of alcohol consumed but also the way people consume it.

While it is clear that prolonged heavy drinking can damage the heart in the ways described above, numerous studies have shown an association

between drinking at more moderate levels and lower risk of CHD in some groups. Epidemiologic studies carried out over the past 25 years suggest that moderate drinking is associated with lower risk of CHD, the top killer in the Nation. A recent study among 22,000 U.S. male physicians, for example, demonstrated that moderate drinking lowers the risk for chest pain and heart attack in apparently healthy men (Camargo et al. 1997). Light-to-moderate drinkers among nearly 86,000 U.S. female nurses showed lower death rates, especially among those at increased risk for CHD, compared with women who did not drink (Fuchs et al. 1995). Data from the National Health Interview Survey (Hanna et al. 1997) and studies in Germany (Keil et al. 1997), China (Yuan et al. 1997), Australia (McElduff and Dobson 1997), and Sweden (Hammar et al. 1997) also found that moderate drinking was associated with lower risk of CHD.

In studies looking specifically at alcohol and heart disease, the term "moderate drinking" has encompassed a wide range of consumption levels, sometimes more than the amount defined by the *Dietary Guidelines for Americans* as moderate: two or fewer standard drinks per day for men and one or less per day for women (USDA 1995*a,b*). However, the apparent benefits of moderate drinking on CHD mortality are offset at higher drinking levels through increasing risk of death from other causes (USDHHS 1999).

Mechanisms. Recent research has suggested several possible mechanisms by which alcohol may protect against CHD. One possibility is that alcohol may impede the accumulation of fatty deposits, or atherosclerotic plaques, in the arteries of the heart by causing changes in a person's cholesterol profile. Researchers have found that alcohol reduced the development of atherosclerotic plaques in the coronary arteries of mice genetically altered to have high levels of LDL-cholesterol—a form of cholesterol that has been found to be associated with increased risk of heart disease (Dai et al. 1997). Furthermore, these animals showed increased levels of HDL-cholesterol, high levels of which are associated with lower risk of CHD.

Other studies have indicated that alcohol consumption increases HDL-cholesterol by decreasing the activity of cholesteryl ester transfer protein (CETP), which transfers cholesterol molecules from HDL particles to LDL or VLDL particles (another form of cholesterol associated with increased risk) (Fumeron et al. 1995). Drinking alcohol was found to alter, at the gene level, the production of two variants of CETP with different activity levels. The change resulted in decreased CETP activity and increased HDL-cholesterol.

These findings provide evidence that one way moderate drinking may lower the risk of CHD is by altering the cholesterol profile. Researchers have confirmed the association between alcohol consumption and increased HDL-cholesterol in people who participated in studies in the United States (such as the Framingham Heart Study) (Sonnenberg et al. 1996), France and Ireland (Marques-Vidal et al. 1995), and Finland (Huijbregts et al. 1995).

However, these changes in HDL-cholesterol and LDL-cholesterol levels contribute only about half of the observed protection against CHD with alcohol consumption. Researchers are therefore investigating alcohol's anti-blood clot (antithrombotic) effect. Blood platelets and clotting factors cause blood clots or thrombi to form in coronary arteries narrowed by atherosclerosis, thereby precipitating heart attacks. Researchers have found that alcohol consumption is associated with antithrombotic effects, such as reduced platelet activation and clotting factor activity (Rubin and Rand 1994).

Several studies suggest that alcohol acts on clot-related proteins in the blood. For example, one study found that drinking 30 grams of alcohol (about two and one-half drinks) per day for 4 weeks caused reduced platelet aggregation and decreased blood levels of fibrinogen, which stimulates clot formation (Pellegrini et al. 1996). Another study of more than 600 physicians found a positive association between moderate alcohol intake and blood levels of tissue plasminogen activator (tPA), an enzyme that breaks down blood clots (Ridker et al. 1994). In a laboratory

study, cultured endothelial cells, which line the artery walls, produced increased levels of tPA with exposure to low levels of alcohol (Aikens et al. 1997). In another recent study, exposure of endothelial cells to low levels of alcohol suppressed the production of substances that promote clotting and stimulated the production and activity of substances (such as tPA) that inhibit clotting (Booyse 1999).

Moderate alcohol consumption may also lower CHD mortality by improving survival after a heart attack (Dufour et al. 1996; Wannamethee et al. 1995). For example, a recent analysis of more than 14,000 subjects followed over 20 years in the National Health and Nutrition Examination Survey Epidemiologic Followup Survey found that regular drinkers are more likely to survive myocardial infarction than abstainers are (Dufour et al. 1996). Although epidemiologic data regarding the association between alcohol consumption and improved heart attack survival are still limited, researchers are already discovering some of the cellular mechanisms by which alcohol may have such an effect.

Researchers have known for some time that alcohol increases blood levels of the chemical adenosine, which protects or "preconditions" heart cells against damage. Transient blockage of a coronary artery can induce this preconditioning by adenosine; administering compounds known to increase adenosine—alcohol is one—also has this effect. Investigators recently found that hearts from guinea pigs fed alcohol for 3 to 12 weeks had less damage and greater recovery of their ability to contract after an experimentally induced heart attack (Miyamae et al. 1997). They also found that blocking adenosine docking sites or receptors on the surfaces of myocytes inhibited alcohol's protective effect. Another recent study demonstrated that hearts from rats already preconditioned by brief ischemia prior to an experimental heart attack could be further protected if the animals had been chronically consuming alcohol (McDonough 1997).

The intracellular mechanisms of alcohol's apparent protective effect against heart attack injury activated by adenosine remain unclear. Researchers are examining several possibilities, such as activation of the myocyte enzyme protein kinase C, which may beneficially alter movement of ions (electrolytes) through the myocyte surface that are key to cell function and contractility (Rodriguez et al. 1998); increased production of protective compounds such as nitric oxide by endothelial cells (Davda et al. 1993); and activation of molecules (transcription factors) that turn on genes responsible for producing protective enzymes and proteins (Zeldin et al. 1996). Further studies will be needed to understand these intracellular mechanisms and to determine their applicability in humans.

Alcohol and Stroke

The relationship between alcohol consumption and stroke is similar to that seen with CHD. Studies have found that while moderate alcohol consumption is associated with lower incidence of ischemic strokes, in which the blood supply to the brain is blocked, heavy drinking may increase the risk of both ischemic and hemorrhagic strokes (Palomaki and Kaste 1993; Stampfer et al. 1988). The mechanisms behind any apparent protection by moderate alcohol intake against ischemic stroke are likely to be the same as with CHD—for example, improved cholesterol levels and antithrombotic effects, which decrease the likelihood of atherosclerosis and thrombus formation in the arteries of the brain. Future studies should also consider the potential detrimental effects of alcohol withdrawal, since stroke may occur in alcoholics during the withdrawal period.

Alcohol and Blood Pressure

An association between heavy alcohol consumption and increased blood pressure (hypertension) has been observed in more than 60 studies in diverse cultures and populations (recent studies include Ascherio et al. 1996; Seppa et al. 1996; York and Hirsch 1997). It is clear that heavy alcohol consumption elevates blood pressure, causing or exacerbating hypertension. However, controversy remains as to whether moderate

alcohol consumption has any beneficial effects on blood pressure.

Deleterious Effects of Alcohol on Blood Pressure. Heavy alcohol consumption reversibly increases blood pressure in people with and without hypertension (Puddey et al. 1995; Ueshima et al. 1993). It is estimated that one drink a day can chronically increase blood pressure 1 millimeter mercury in middle-aged individuals, and even more in the elderly and in people who already have high blood pressure (Beilin et al. 1996). This raises the possibility that regulating alcohol intake might be one means of reducing blood pressure in people with hypertension. A 2-year study found that hypertensive drinkers could reduce their blood pressure after educational intervention and through self-regulation of alcohol intake (Lang et al. 1995). Given the abundance of data associating alcohol with hypertension, the World Health Organization and the International Society of Hypertension have jointly recommended reducing daily alcohol intake to treat high blood pressure (World Health Organization 1996).

Recently, the Prevention and Treatment of Hypertension Study looked at the effectiveness of a 6-month behavior intervention program to reduce both alcohol intake and blood pressure among drinkers who consumed at least three drinks daily and who had normal to slightly elevated blood pressure (Cushman et al. 1998). After 6 or more months, individuals in the behavioral intervention group consumed approximately 1.3 fewer drinks per day than those in the control group. This reduction in alcohol consumption did not result in a significant effect on blood pressure. The authors concluded that although greater reductions in alcohol intake might have led to greater reductions in blood pressure, the study results did not strongly support reducing alcohol consumption below two drinks daily as a sole means of preventing or treating hypertension.

Despite the well-recognized association between alcohol and hypertension, the cellular mechanisms of alcohol's effect on blood pressure remain uncertain. Especially confusing is the fact that, initially, drinking alcohol dilates blood vessels, which lowers blood pressure. Studies looking to explain how long-term, heavy alcohol consumption reverses this lowering and leads to elevated blood pressure suggest effects by alcohol on the autonomic nervous system, an important regulator of blood pressure. For example, heavy alcohol consumption has been associated with increased release of the stress hormones adrenaline and noradrenaline (reflecting activation of the sympathetic component of the autonomic nervous system). This release causes constriction of blood vessels—and hence increased blood pressure—and decreased sensitivity of sensory neurons called baroreceptors, which send signals from the heart and large arteries to the brain to regulate blood pressure.

In addition to its effects on central regulation of blood pressure, heavy alcohol consumption also may alter peripheral regulation of blood pressure by affecting smooth muscle cells in the walls of the blood vessels (summarized in Altura and Altura 1996). Chronic alcohol exposure may inhibit the function of endothelial cells, which normally release chemicals to relax the smooth muscle cells in the vessel walls. These vascular cells regulate blood vessel tone and, as a result, blood pressure. Chronic alcohol exposure appears to reduce cellular magnesium levels, which can cause increased calcium fluxes in vascular smooth muscle cells, producing constriction of the blood vessels and increasing blood pressure.

Two studies suggest that increased blood pressure results not from alcohol consumption but from alcohol withdrawal. Investigators found that a single drink of alcohol depressed the blood pressure of patients with hypertension for several hours (Kawano et al. 1996). However, in a related study, the research team found that if patients consumed one drink each evening for 7 days, their blood pressure seesawed, sinking in the evening and rising in the morning (Abe et al. 1994). The studies suggest that regular consumption of alcohol can raise blood pressure during the hours that alcohol is not consumed. These findings are consistent with observations

that sympathetic nerve activity—a regulator of blood pressure—increases during alcohol withdrawal (Denison et al. 1997).

Possible Beneficial Effects of Alcohol on Blood Pressure. Some studies have shown a linear relationship between alcohol and blood pressure at all levels of consumption, whereas other studies have found a J- or U-shaped association, with the lowest levels occurring in people who consumed one to three drinks a day. Most of these studies examined blood pressure among middle-aged people, but a recent study of young adults—aged 18 through 26—found a similar J-shaped relationship between alcohol and blood pressure (Gillman et al. 1995). Taken together with previous data in middle-aged individuals, this suggests that moderate alcohol consumption is associated with a slight reduction in blood pressure or may protect against age-related development of hypertension.

How moderate alcohol consumption might chronically lower blood pressure remains unclear. A recent study found that rats consuming moderate amounts of alcohol for 8 months had lower age-related increases in blood pressure than did animals not given alcohol (Guillaume et al. 1997). The researchers found beneficial changes in kidney receptors for the hormone atrial natriuretic peptide (ANP), which regulates sodium and water levels in the body, and, in turn, blood pressure. Other researchers have found that ANP levels increase with acute alcohol intake (Gianoulakis et al. 1997), promoting water loss and lowering blood pressure. Eight months of moderate alcohol consumption altered kidney ANP receptors in a way that appeared to protect against increasing blood pressure. Future studies will need to clarify the importance of alcohol's influence on ANP in modulating blood pressure, including determining how heavy alcohol consumption may affect ANP receptors and lead to increases in blood pressure.

In a recent study of more than 6,000 people with hypertension, moderate alcohol consumption was associated with lowered mortality due to stroke and heart attacks (Palmer et al. 1995). However, heavy alcohol consumption offsets these effects by increasing the risk of death from causes unrelated to cardiovascular disease. Future studies will be necessary to confirm this finding.

In Closing

Recent research has significantly increased knowledge of how alcohol acts on the heart and the cardiovascular system. Future research will direct the development of therapies to protect against the cardiovascular complications of heavy drinking, although abstinence remains the most likely cure. As important, future studies will elucidate the mechanisms underlying the apparent protective effects of moderate drinking on the heart. Understanding these mechanisms may lead to therapies for patients at risk for myocardial infarction and other cardiovascular events.

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